Chinese Experience in Endovascular Management of Spinal Cord Vascular Malformations

F. LING, T.-L. LI*, Y. BAO**, H. ZHANG, D. WANG

Research Center of Interventional Neuroradiology, Beijing Hospital; Beijing
** Department of Neurosurgery, Pearl River Hospital; Guangzhou*
*** Department of Neurosurgery, The First Hospital Affiliated to the Xingjiang Medical College; Urungi, P.R. China*

Vascular malformations of the spine and spinal cord are uncommon lesions. However they are important clinical entities because they produce considerable morbidity and severely affect the patient's life, family and society. The management of these lesions is difficult. Among modern imaging techniques, selective spinal arteriography plays a decisive role in diagnosis and classification of the spinal vascular malformations. Endovascular embolisation has opened a new treatment for these lesions.

In our series, more than 600 cases of selective spinal angiography were performed from 1984 to 1998. According to the classifications of spinal vascular lesions suggested by Merland, Lasjaunias and others, we classified the 149 cases of spinal vascular lesions into three groups:

- Group 1. This group is defined as intracanal vascular malformations. They are subclassified into intramedullary arteriovenous malformations, perimedullary arteriovenous fistulas, and spinal dural arteriovenous fistulas with medullary drainage.
- Group 2. This group consists of vertebral vascular lesions, mainly hypervascular tumours, including haemangioma, aneurysmal bone cyst, haemangiopericytoma, and metastatic tumours etc.

Table 1 **Radial Pressures: peak systole / peak diastole**

Group I	Intramedullary AVM	76 cases
	Perimedullary AVF	51 cases
	Spinal dural AVF	39 cases
Group II	Vertebral haemangioma	5 cases
	Haemangiopericytoma	1 case
	Metastatic tumour	5 cases
Group III	Paravertebral AVF	1 case
	(Cobb's syndrome)	14 cases

- Group 3. This group consists of paravertebral arteriovenous fistulas and metameric angiomatosis, such as Cobb's syndromes, Osler-Weber-Rendy syndromes, etc.

A satisfactory outcome can only be achieved by the best therapeutic modality according to the classification of the lesion. In our series of 172 cases treated, 74 cases are excellent, 72 cases are good, 18 fair, 8 worse. Using the classic outcome scale, four grades were defined:

Excellent. The patient completely recovers with complete obliteration of the malformation, or more than 90% of the malformation embolized.

Good: The patient improves with more than 50% of the malformation embolized.

Fair: The patient remains stable with some reduction of the malformation.

Worse. The patient becomes worse with permanent residual neurological deficit and no reduction of the AVM.

In endovascular therapy of the spinal vascular lesions, several microcatheter systems listed below are often used:

1. TRACKER-10, 18 microcatheters and their compatible guidewires (Target Co. USA).

2. TRANSIT microcatheters and guidewires (Cordis Co. USA).

3. JETSTREAM microcatheters (MIS Co. USA).

4. MAG 3F/2F microcatheters (Balt Co. France).

Embolic agents

1) NBCA (N-butyl-2-cyanoacrylate). It is often used in low concentration (25%~30%) and small amounts (0.08~0.15 ml) with slow injection inside the nidus.

2) EMBOSPHERE (Guerbet Co. France). They are gelatum microspheres with a hydrophilic membrane. The Embosphere ranging from 500 to 700 micron in diameter is often used. Small amounts with slow injection may effectively occlude the malformation nidus.

3) PULSAR (MIS Co. USA). They are opaque microspheres with a hydrophilic membrane and tantalum powder.

4) Microballoons (Balt Co. France). They are only used for some straight gross feeders and huge fistulas.

5) Microcoils and microspirals (platinum or tungsten coils and spirals, MDS or GDC. They are often used for some fistulas.

6) Polyvinyl alcohol foam (PVA), silk thread, dura mater were only used by us in the early period. Now, they are seldom adopted.

Section 1 Intramedullary Arteriovenous Malformations

Intramedullary AVMs are defined as spinal vascular lesions in which the niduses of the AVM are within the parenchyma of the spinal cord or pia, receiving their blood supply from the spinal arteries. Intramedullary AVMs are subclassified into glomus AVMs, consisting of compact niduses of abnormal vessels within the spinal cord, and juvenile AVMs, consisted of a much looser tangle of abnormal vessels that occupy almost the entire spinal canal at the involved level.

The angioarchitecture of the intramedullary AVMs is similar to that of the cerebral AVMs. Due to the fact that the blood supply may be in terminal form or en passage form, the selection of embolisation approaches and embolic agents should be cautiously considered. Aneurysms and dilation of the medullary veins often accompany the intramedullary AVMs and are usually responsible for subarachnoid haemorrhage. The existence of the aneurysm and dilation of the medullary veins is helpful in predicting the clinical presentations and prognosis.

Intramedullary AVMs frequently occur in adolescence or younger adult. In our series of 76 cases, 27% of the patients were under 30 years old. The most common clinical presentations are bleeding (subarachnoid haemorrhage or intramedullary haematoma) accounting for 31%, progressive myelopathy accounting for 63%. 70 per cent of the lesions are located in the cervical and lumbar enlargement of the spinal cord.

59.2% of the intramedullary AVMs were treated with embolisation only, 28.9% with a combination of embolisation and surgery, 11.8% with surgery only. The outcome of the 76 patients we treated showed 21 cases (27.6%) were cured, 19 cases (25.0%) were improved remarkably, 23 cases (30.2%) were fair, 8 (10.5%) were unchanged and 5 cases (6.5%) were worse.

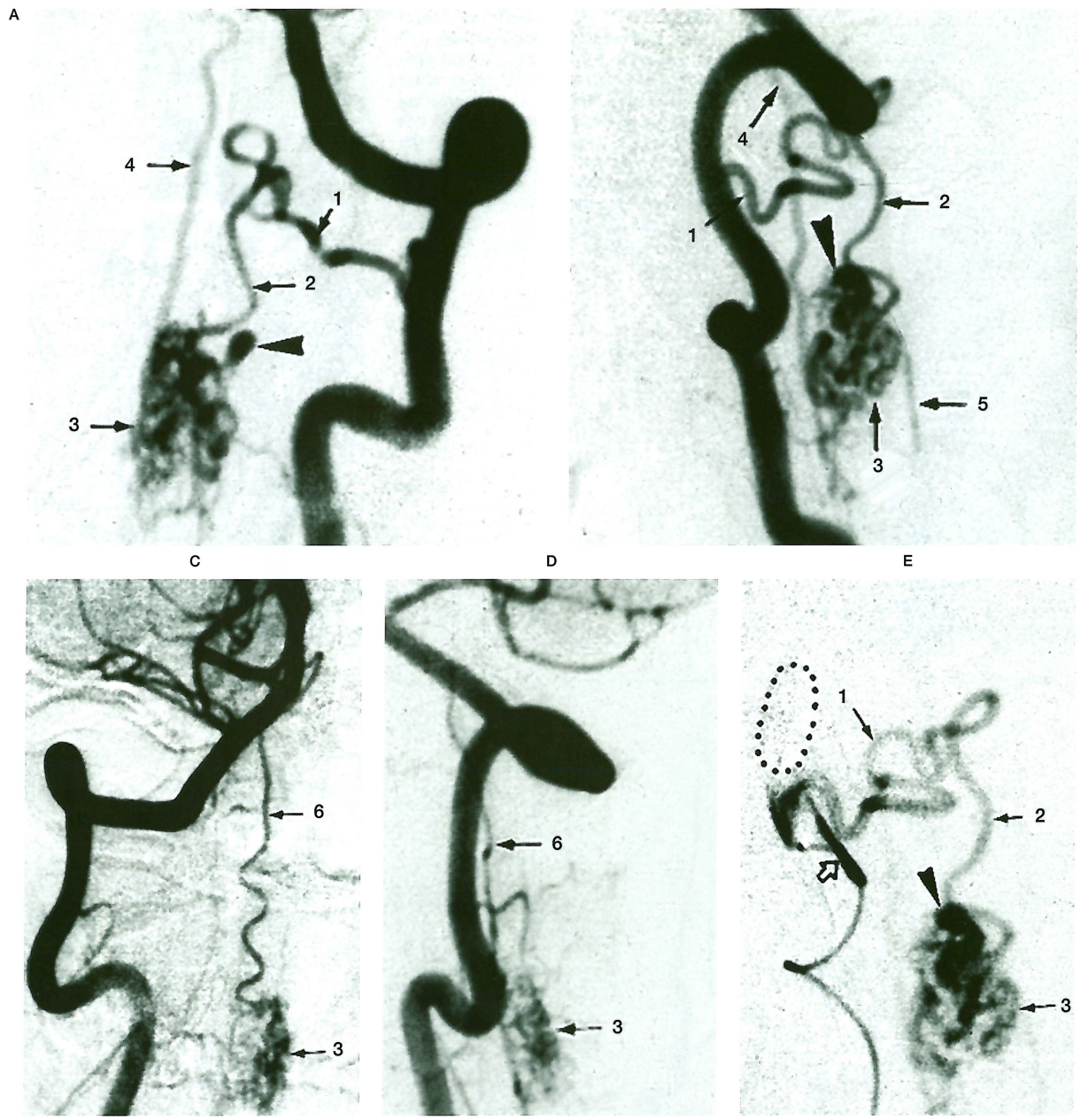
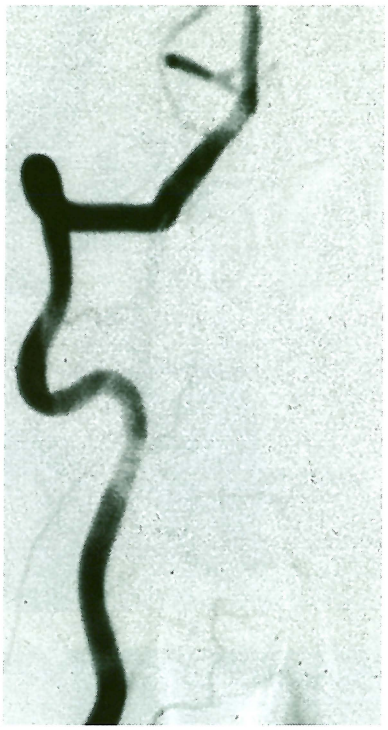


Figure 1 Cervical juvenile-type intramedullary AVM. Embosphere embolisation with temporary occlusion of the vertebral artery. A 23-year-old male presented with a history of acute onset of paraplegia, dyspnea, and sphincter disturbance. The patients muscle power improved two months later. A, B) Left vertebral angiograms: (A) anteroposterior and (B) lateral projections show an intramedullary AVM (3) supplied by a posterior spinal artery (2) of the posterior radiculo-medullary artery (1) originating from the left vertebral artery at the level of C2. The venous drainage is to the anterior (4) and posterior medullary veins (5). Arrowhead marks an aneurysm. C, D) Right vertebral angiograms: (C) anteroposterior and (D) lateral projections, show the anterior spinal artery (6), originating from the vertebral artery under the origin of posterior inferior cerebellar artery, also contributing to the malformation (3). E) Because the posterior radicular artery (1) was long and twisted, the Tracker- 18 microcatheter (Target Co.) could not get nearer to the malformation (3). In order to prevent embolic agent migrating to the posterior cerebral circulation, a non-detachable balloon catheter (Magic B1, Balt Co.) was introduced and temporarily occluded the vertebral artery distal to the orifice of the feeding artery with systematic heparinization. The malformation was embolised through slow injection of Embosphere (500~700 μm) with repeated control angiography until the nidus disappeared. Note the contrast media retained (open arrow) in the vertebral artery because of reflux. After embolisation, the Tracker-18 microcatheter was withdrawn and the lumen of the vertebral artery was repeatedly irrigated with saline. Then the balloon catheter the balloon inflated was gently pulled back and drove potential residual embolic agent into the subclavian artery.

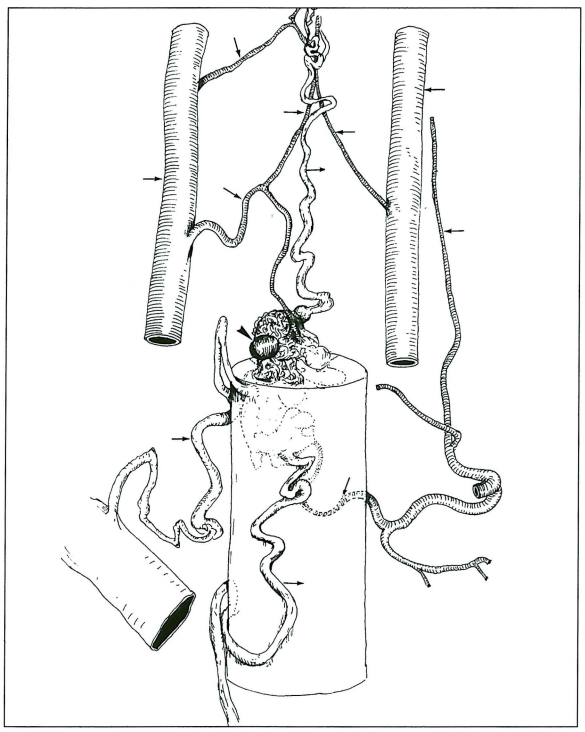
F

Figure 1 F-I) Left vertebral angiograms after embolisation, (F) anteroposterior and (G) lateral projections, show complete obliteration of the malformation. It is interesting that the malformation was also not opacified in the angiogram of (H) the right vertebral artery from which embolisation was not performed. The patient's symptoms have remarkably improved. I) Artist's drawing of the AVM angioarchitecture (anterior view).

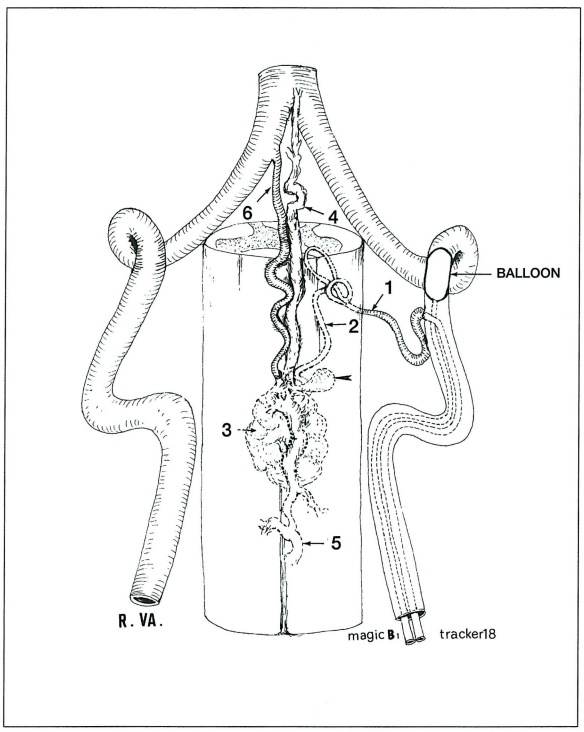
H



G



I



Comment – *In this cervical juvenile-type intramedullary AVM, all feeding arteries contribute to the nidus of AVM which presented as a single compartment. After particulate embolisation which effectively packed the whole nidus via the relatively approachable feeding artery, angiography through other feeding arteries did not opacify the nidus.*

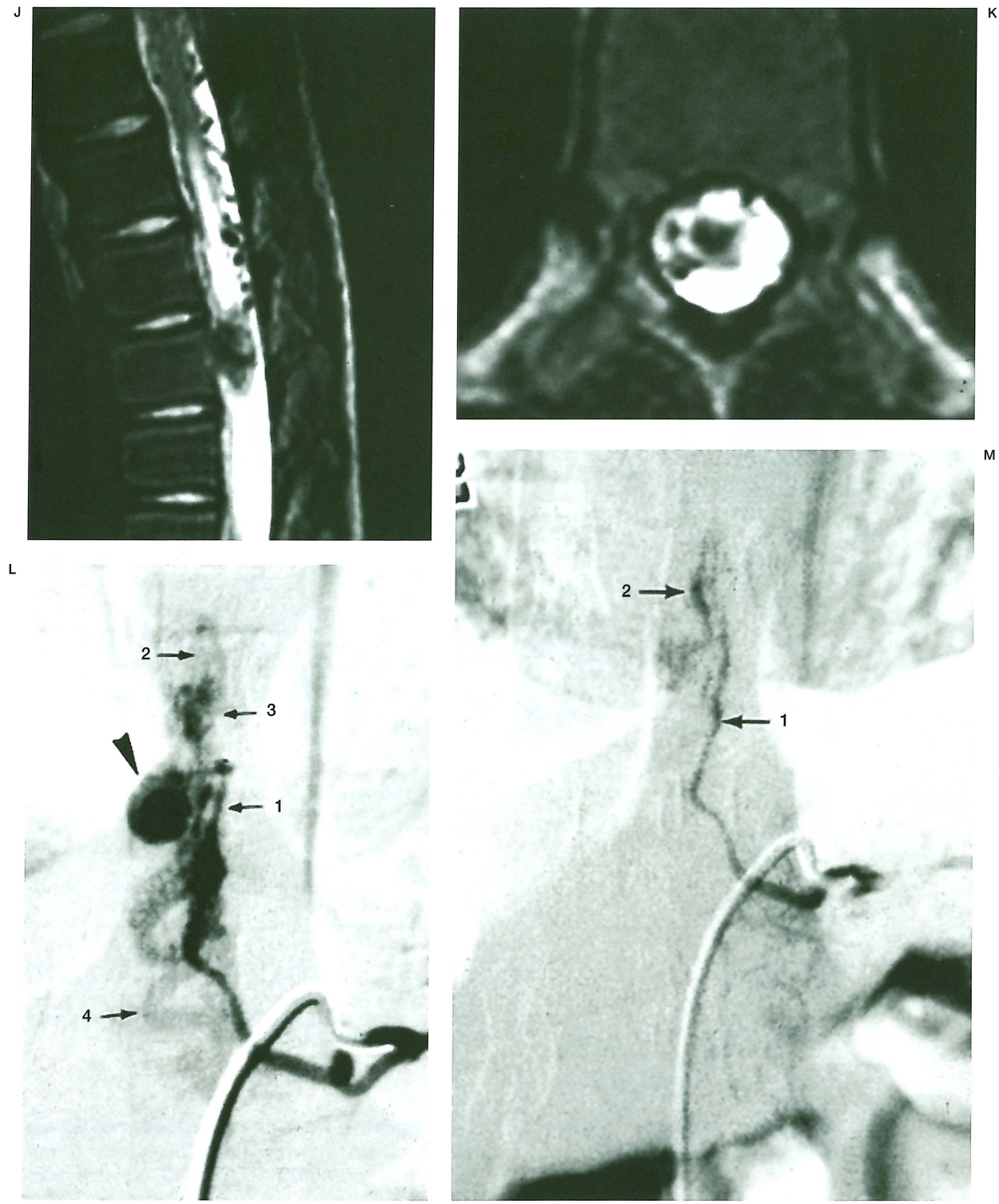
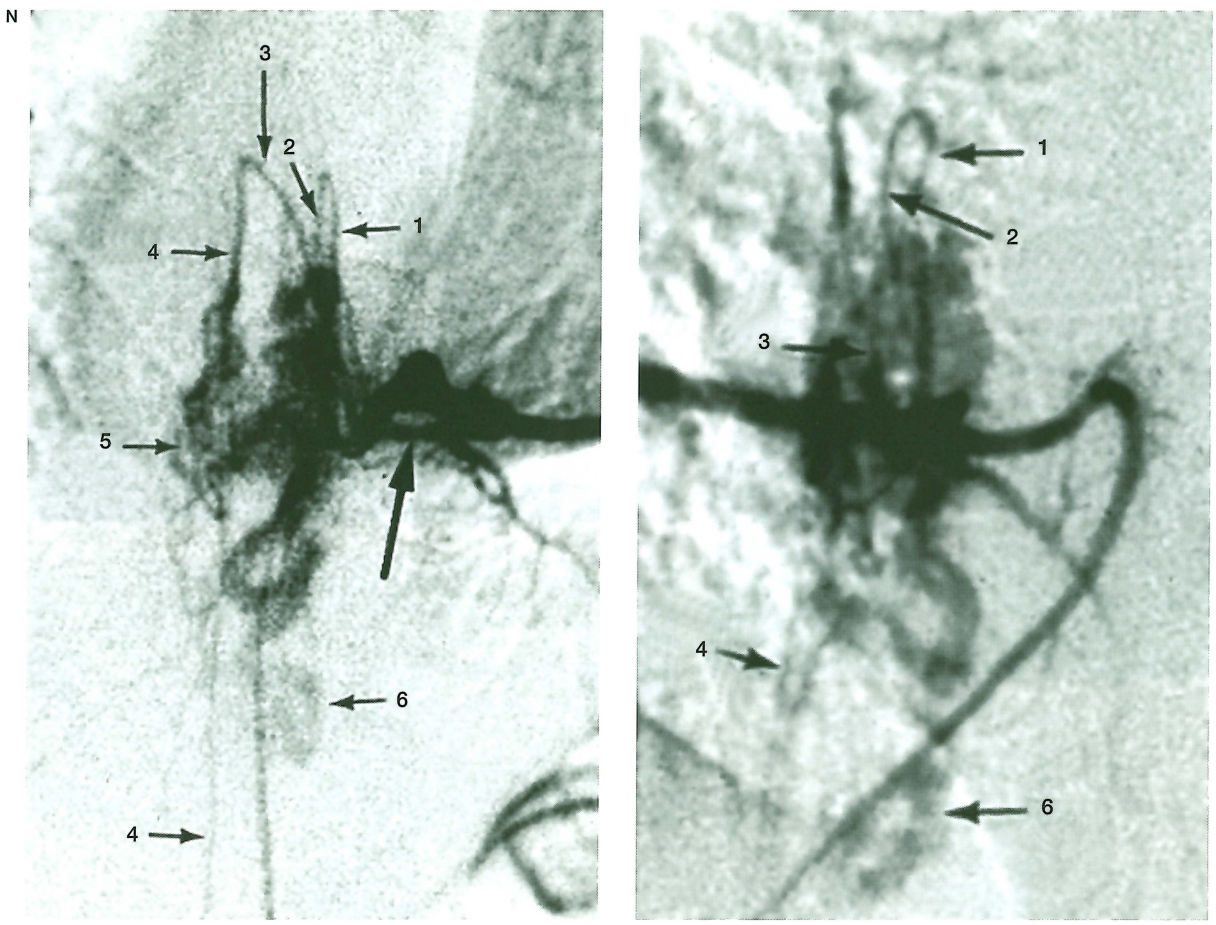


Figure 1 J-M) T8-9 intramedullary AVM with an aneurysm. A 16-year-old male presented with acute onset of paraparesis with grade 2-3 muscle power for 20 days. (J) Sagittal and (K) axial T2-weighted MR images show the intramedullary signal void at the T8-9 level. L) Selective injection of the left T10 intercostal artery in A-P view shows an AVM (3) supplied by the posterior spinal artery (2) of the posterior radiculo-medullary artery (1) with caudal venous drainage (4). Note the aneurysmal dilation (arrowhead). M) Angiogram of the left T10 intercostal artery after embolisation with Embosphere (500-700 μm) shows complete obliteration of the nidus, aneurysm and the draining vein, with preservation of the posterior spinal artery (2).



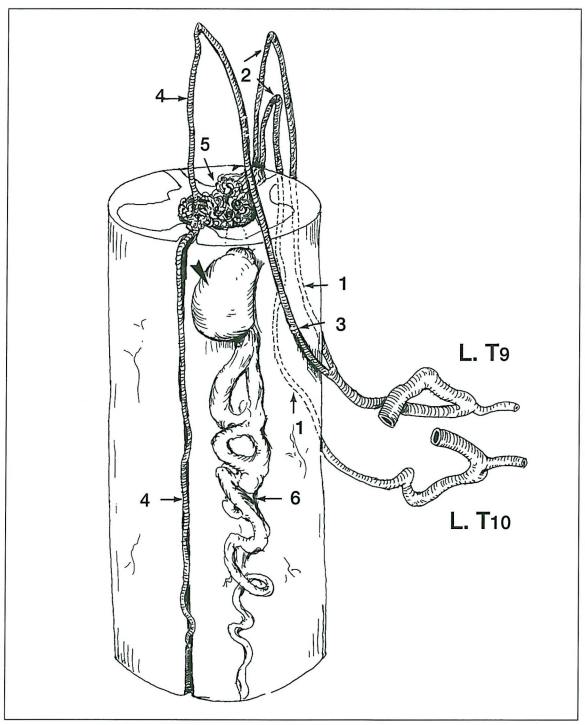


Figure 1 N-O) Selective injections of the left T9 intercostal artery, (N) frontal and (O) lateral projections, show a radiculo-medullary trunk (arrow) dividing into the anterior (3) and posterior radiculo-medullary artery (1) and then the anterior spinal axis (4) and posterior spinal artery (2), contributing to the malformation (5) with the same drainage as in figure 1L (6). Because the anterior spinal axis (4) supplies the malformation in an indirect manner with its distal branch supplying the caudal part of the spinal cord, embolisation was contraindicated.
P) Artist's drawing of the AVM angioarchitecture (anterior view). 1) Posterior radiculomedullary artery; 2) Posterior spinal artery; 3) Anterior radiculomedullary artery; 4) Anterior spinal artery; 5) AVM nidus; 6) Draining vein. Arrowhead: Venous pouch.

Comment – *In this intramedullary AVM, the aneurysm arising from the posterior spinal artery was probably responsible for haemorrhage. If microcatheter could have gone further into the aneurysm and nidus, they could have been embolised more efficaciously with NBCA to prevent further bleeding except that the residual nidus was still supplied by the left T9 intercostal artery.*

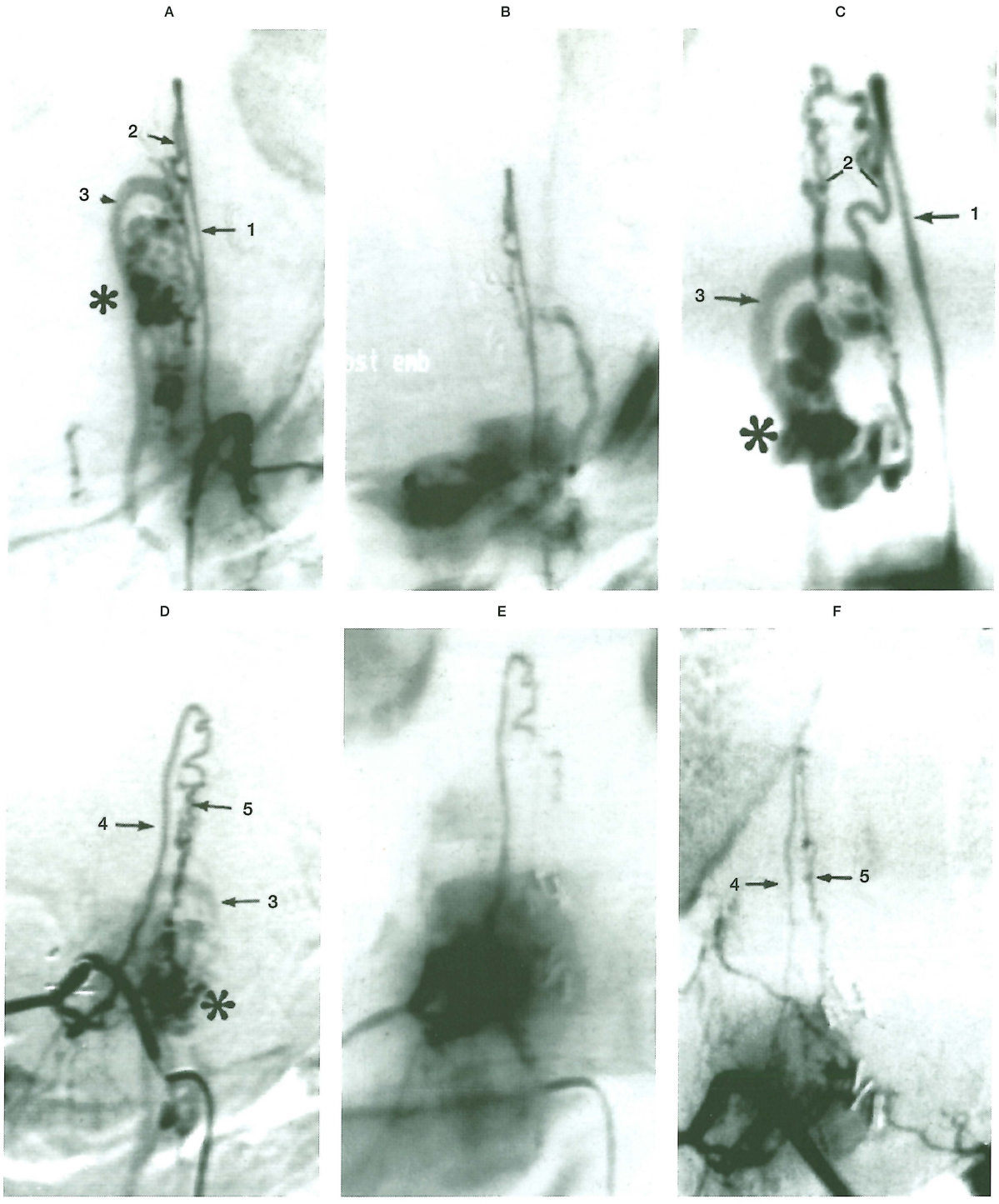


Figure 2 Conus medullaris juvenile-type intramedullary AVM. A 22-year-old male presented with acute onset of paraplegia and lumbodorsal pain for one month. A-C) Selective injections of the left L1 lumbar artery in A-P views. A) Pre-embolisation angiogram shows the nidus (asterisk) supplied by a posterior spinal artery (2) arising from the posterior radiculomedullary artery (1) with a caudal draining vein (3). B) Angiogram after PVA embolisation demonstrates complete obliteration of the nidus. C) Angiogram four years after embolisation shows partial recanalization of the nidus (asterisk) and a newly developed posterior spinal artery (2) contributing to the nidus. D-F) Selective injections of the right T12 intercostal artery in A-P views. D) Pre-embolisation angiogram shows another posterior spinal artery (5) arising from the radiculo-medullary artery (4) contributing to the same AVM (asterisk) with the same draining vein (3). E) Postembolisation angiogram shows complete obliteration of the nidus. F) Follow-up angiogram four years later demonstrates no recanalization.

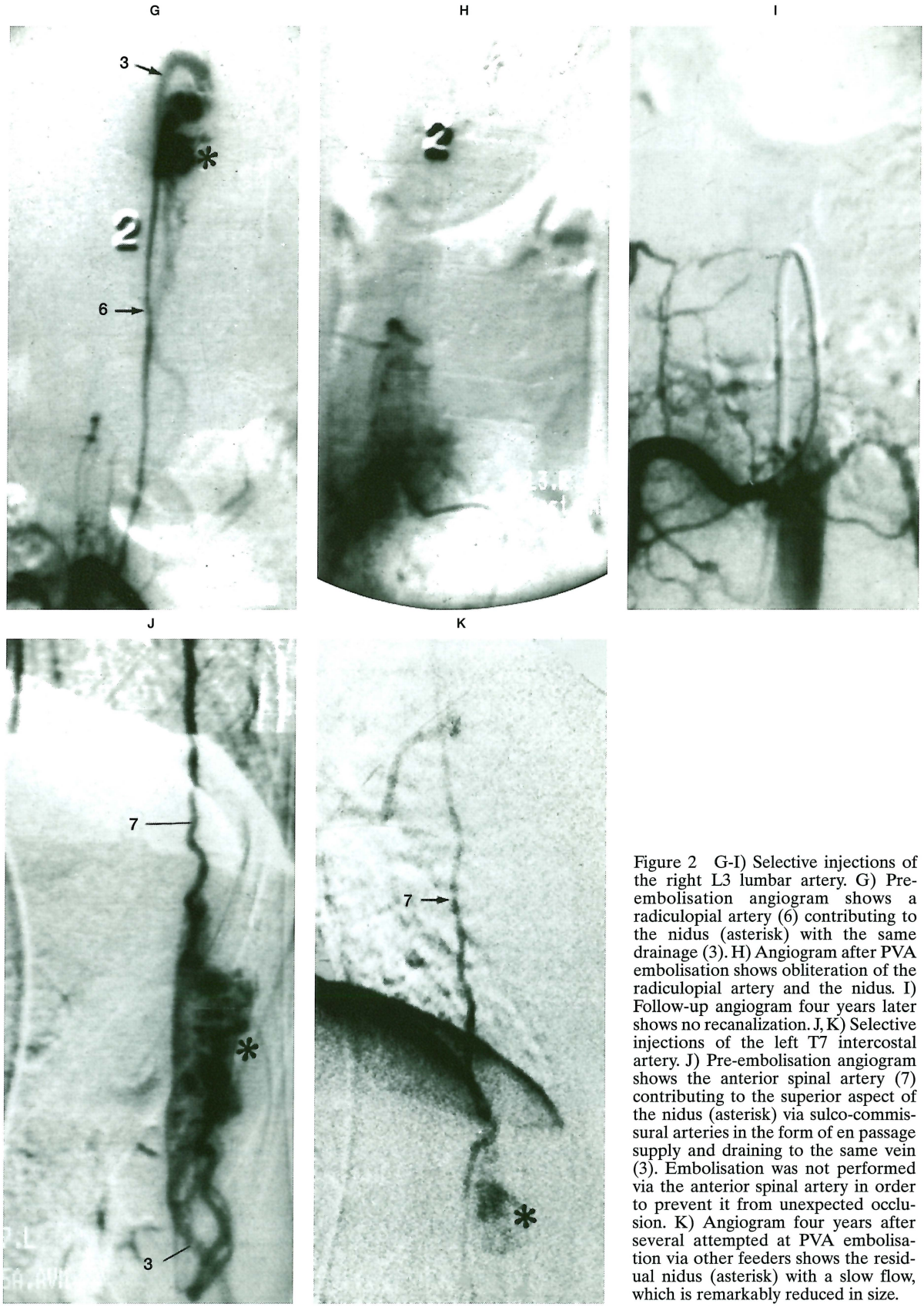


Figure 2 G-I) Selective injections of the right L3 lumbar artery. G) Pre-embolisation angiogram shows a radiculopial artery (6) contributing to the nidus (asterisk) with the same drainage (3). H) Angiogram after PVA embolisation shows obliteration of the radiculopial artery and the nidus. I) Follow-up angiogram four years later shows no recanalization. J, K) Selective injections of the left T7 intercostal artery. J) Pre-embolisation angiogram shows the anterior spinal artery (7) contributing to the superior aspect of the nidus (asterisk) via sulco-commissural arteries in the form of en passage supply and draining to the same vein (3). Embolisation was not performed via the anterior spinal artery in order to prevent it from unexpected occlusion. K) Angiogram four years after several attempted at PVA embolisation via other feeders shows the residual nidus (asterisk) with a slow flow, which is remarkably reduced in size.

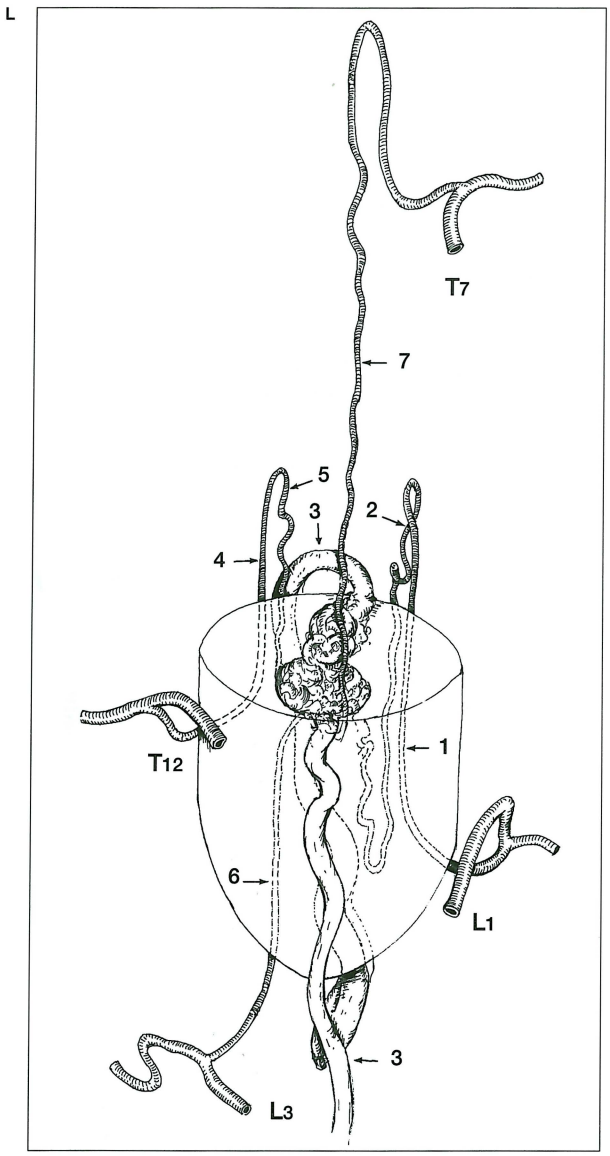


Figure 2 L) Artist's drawing of the AVM angioarchitecture (anterior view).

Comment – *The patient's lumbar pain was completely relieved immediately following the first embolisation via the three posterior spinal arteries. Ten days later he was able to stand and then completely recovered six months later. The angiography four years after embolisation demonstrated that the AVM had markedly decreased in size. During a ten-year follow-up, no neurological deterioration has occurred, indicating that the embolisation through the posterior spinal arteries can reduce the stealflow and improve the spinal circulation and neurological functions.*

Section 2 Perimedullary Arteriovenous Fistulas

The perimedullary arteriovenous fistulas (AVFs) are direct arteriovenous shunts without a nidus of abnormal vessels between the spinal arteries and medullary veins. Although some authors categorized the lesion as a spinal arteriovenous malformation, we think that the perimedullary AVFs have their own individual characteristics in angioarchitecture and can be divided into three types according to the size of the fistulas. It is useful in selection of therapeutic modalities and in prediction of prognosis.

- Type I. A small low-flow single-hole fistula without obvious dilatation of the feeding artery and draining vein. The fistula is at the point where there is a change in vascular calibre.
- Type II. A high-flow single-hole fistula with apparent dilatation of the feeding artery and draining vein. There often is an arterialized venous pouch near the fistula.
- Type III. A giant high-flow fistula with multiple feeders and a highly dilated vein. The varix often occupies the whole canal at the fistula level and the fistula site is hard to localize.

The clinical presentations are similar to those of intramedullary AVMs, including bleeding, progressive myelopathy, and radicular pain etc. The lesions frequently occur in the second or third decades. In one third of the cases, these patients had an overlooked history of meningitis during childhood. The lesions may occur at any level of the spinal cord. If a proper therapeutic modality can be chosen according to their type of the lesions, the cure rate will be higher.

In our series of 51 patients with the perimedullary AVFs, 16 cases had Type I, 19 cases Type II, and 16 cases Type III. Type II and III AVFs are suitable for embolisation and Type I for surgery. Pre-operative embolisation of the dominant feeders in Type II will reduce the venous pressure and make the operation easy. The outcome of the patients we treated showed 27 cases were excellent, 17 cases were good, 4 cases were fair, 3 cases had neurological complications.

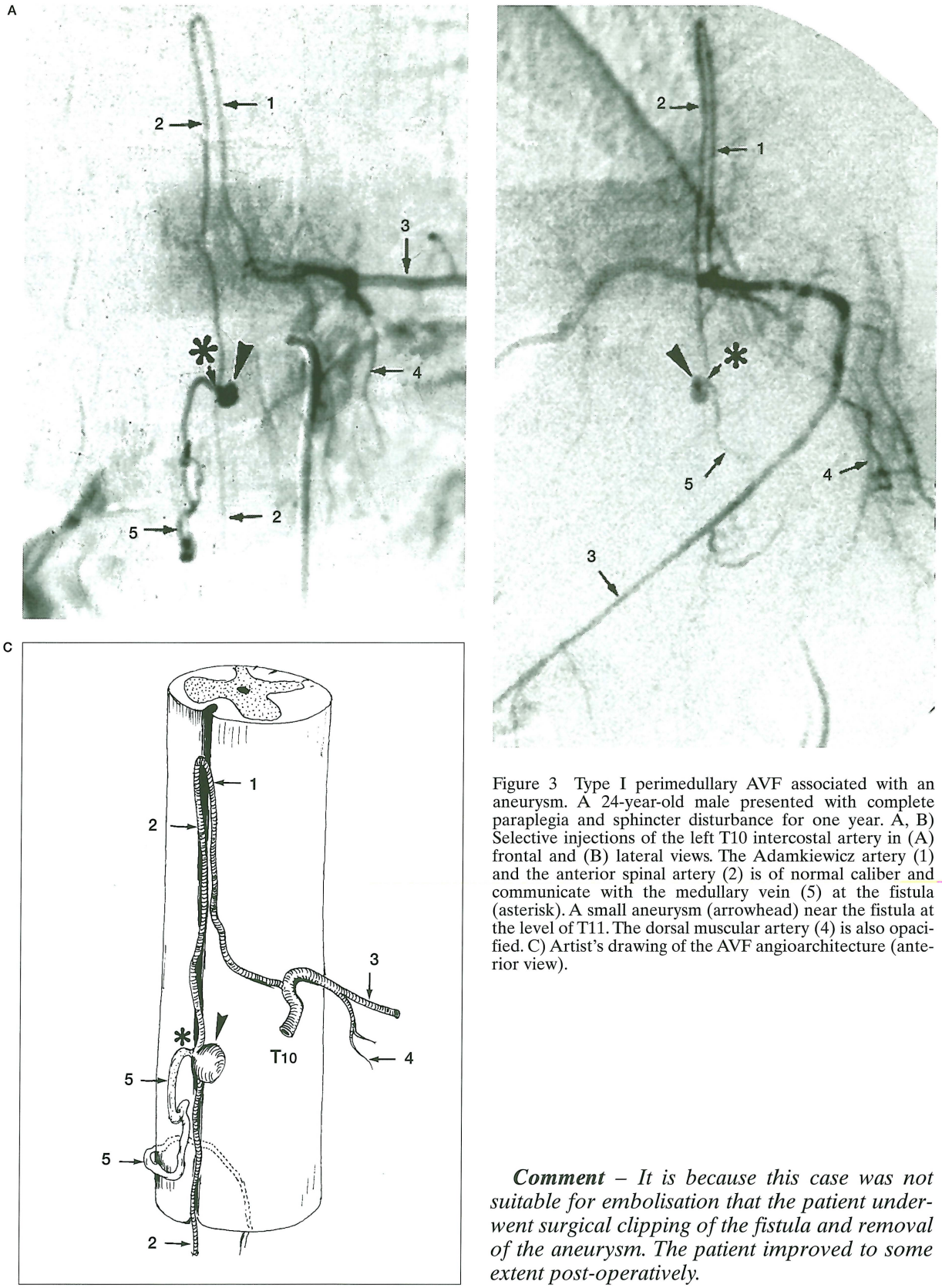


Figure 3 Type I perimedullary AVF associated with an aneurysm. A 24-year-old male presented with complete paraplegia and sphincter disturbance for one year. A, B) Selective injections of the left T10 intercostal artery in (A) frontal and (B) lateral views. The Adamkiewicz artery (1) and the anterior spinal artery (2) is of normal caliber and communicate with the medullary vein (5) at the fistula (asterisk). A small aneurysm (arrowhead) near the fistula at the level of T11. The dorsal muscular artery (4) is also opacified. C) Artist's drawing of the AVF angioarchitecture (anterior view).

Comment *– It is because this case was not suitable for embolisation that the patient underwent surgical clipping of the fistula and removal of the aneurysm. The patient improved to some extent post-operatively.*

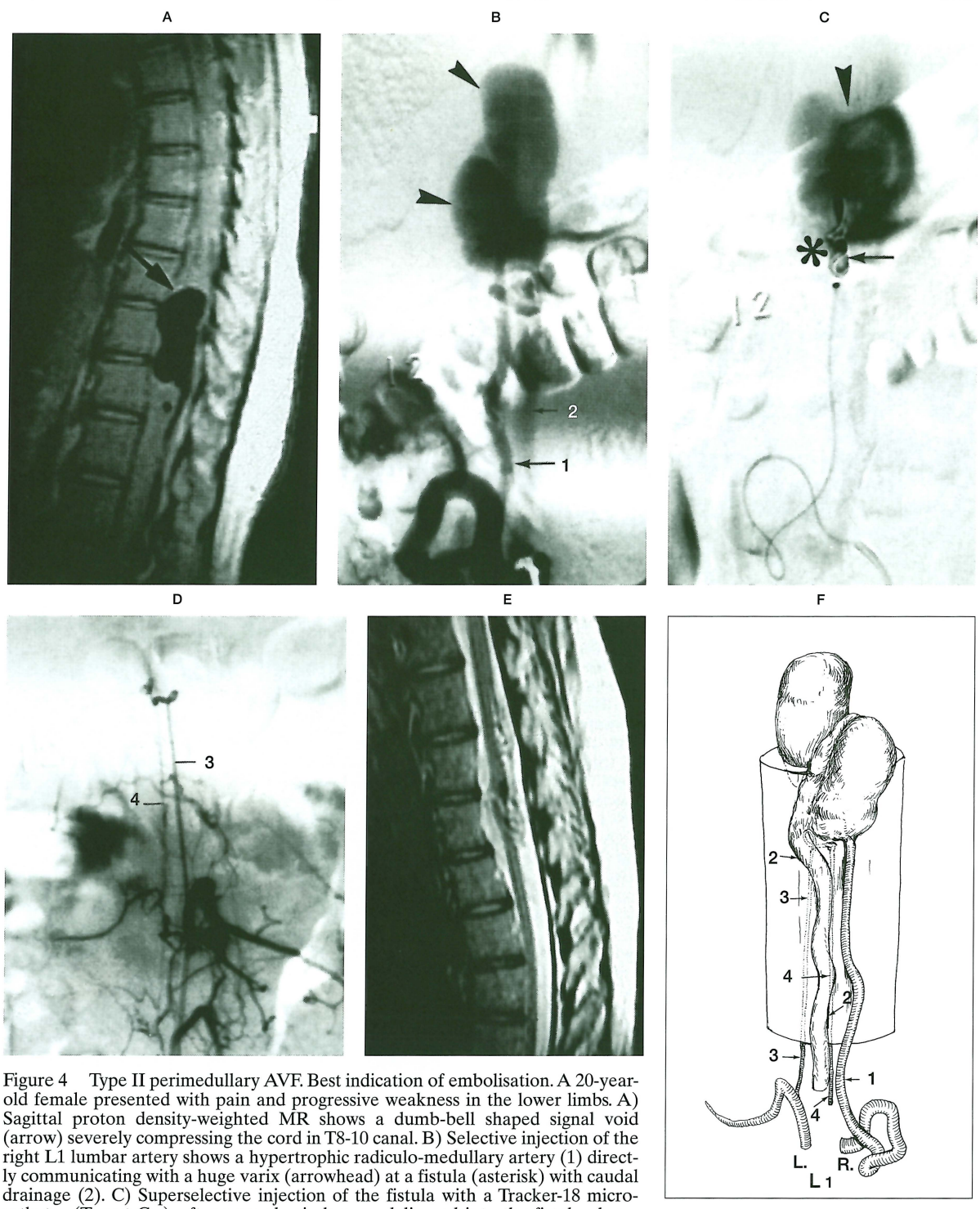
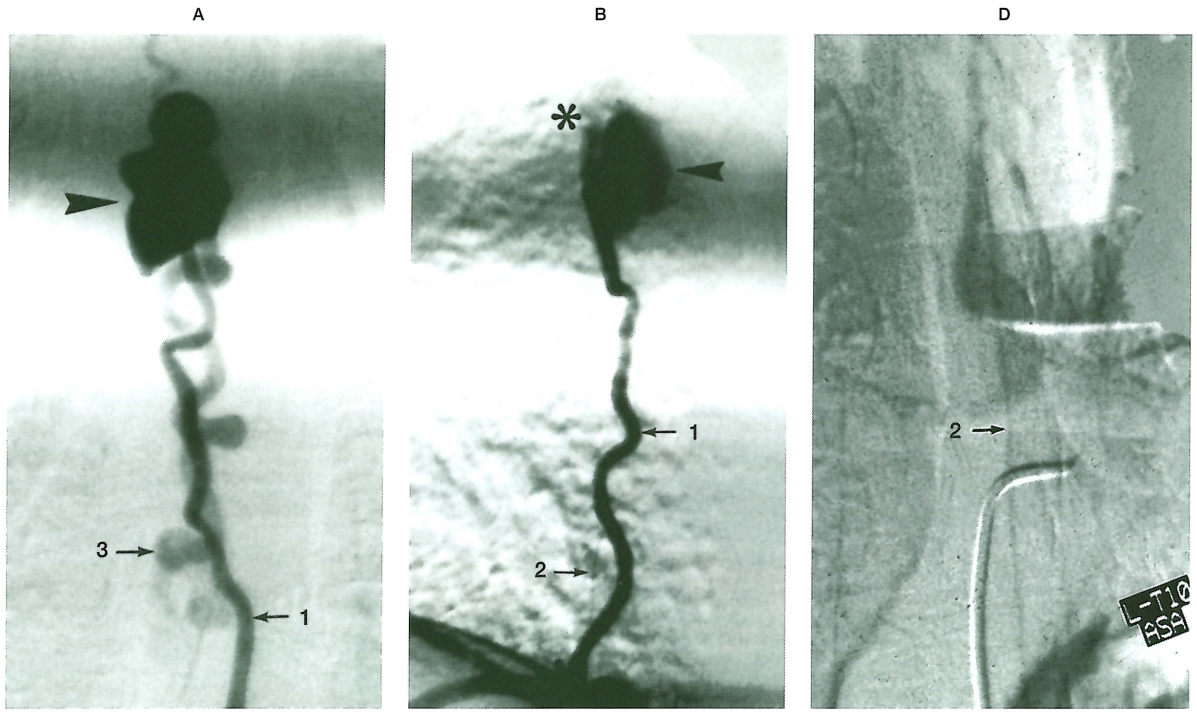
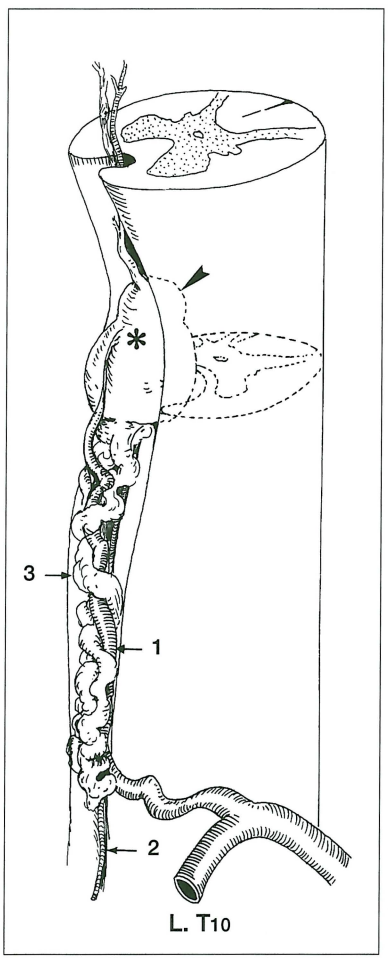


Figure 4 Type II perimedullary AVF. Best indication of embolisation. A 20-year-old female presented with pain and progressive weakness in the lower limbs. A) Sagittal proton density-weighted MR shows a dumb-bell shaped signal void (arrow) severely compressing the cord in T8-10 canal. B) Selective injection of the right L1 lumbar artery shows a hypertrophic radiculo-medullary artery (1) directly communicating with a huge varix (arrowhead) at a fistula (asterisk) with caudal drainage (2). C) Superselective injection of the fistula with a Tracker-18 microcatheter (Target Co.), after several spirals were delivered into the fistula, shows nearly complete occlusion of the fistula (asterisk), the varix (arrowhead) and the embolic spirals (arrow). D) Postembolisation selective injection of the left L1 lumbar artery shows the anterior radiculo-medullary artery (3) filling the anterior spinal axis (4) without opacification of the varix. E) Sagittal MR image nine months after embolisation shows the varix shrunk. The hyperintensity signal inside the varix is due to thrombosis. The diameter of the spinal cord has returned to normal size. F) Artist's drawing of the AVF's angioarchitecture (posterior view).

Comment – *Type II perimedullary AVF, commonly supplied by huge and straight feeding arteries with high flow, is most suitable for endovascular embolisation.*





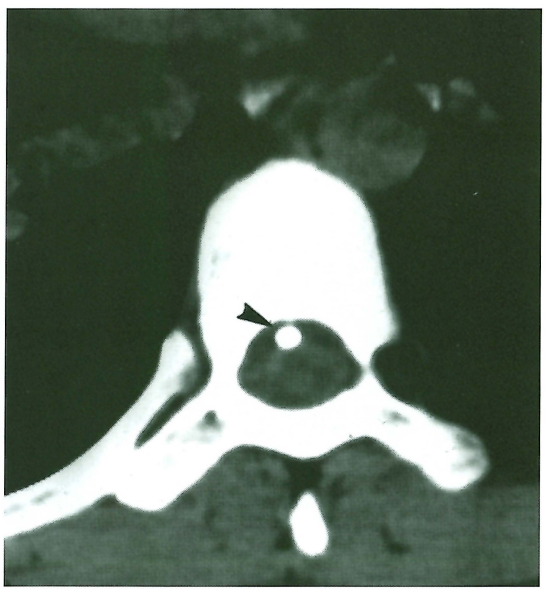


Figure 5 Type III perimedullary AVF treated with detachable balloon embolisation. An 18-year-old male presented with weakness and atrophy of the right leg for ten years. A, B) Selective injections of the left T10 intercostal artery in (A) anteroposterior and (B) lateral projections show a varix (arrowhead) supplied by the markedly dilated anterior radiculomedullary artery (1) at a fistula (asterisk) in the anterior margin. The draining vein (3) is tortuous and dilated. The anterior spinal axis (2) is also opacified. C) The fistula was occluded with a detachable balloon via the anterior spinal artery (2). CT scan two weeks after embolisation shows the detachable balloon (arrowhead) in the ventral subarachnoid spaces. D) Selective injection of the left T10 intercostal artery six months after embolisation shows complete obliteration of the varix and the intact anterior spinal axis (2). The patient completely recovered. E) Artist's drawing of the AVF's angioarchitecture (lefl anterolateral view).

Comment – *Sometimes perimedullary AVFs are supplied by straight and greatly dilated arterial feeders, they allow placement of a detachable balloon at the fistula site with complete anatomic and clinical cure. However, the use of detachable balloons is limited by the tortuosity of the feeding arteries.*

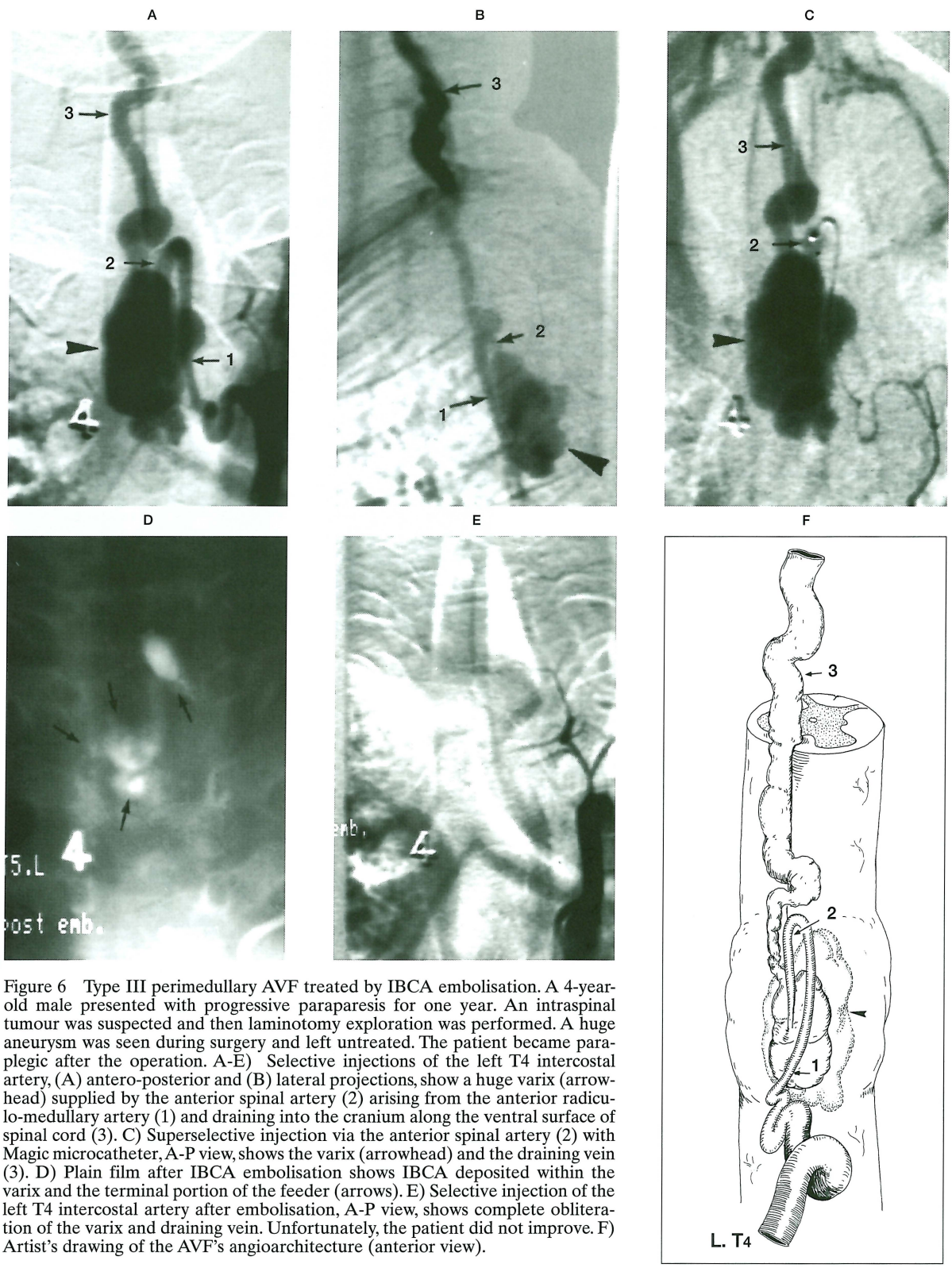


Figure 6 Type III perimedullary AVF treated by IBCA embolisation. A 4-year-old male presented with progressive paraparesis for one year. An intraspinal tumour was suspected and then laminotomy exploration was performed. A huge aneurysm was seen during surgery and left untreated. The patient became paraplegic after the operation. A-E) Selective injections of the left T4 intercostal artery, (A) antero-posterior and (B) lateral projections, show a huge varix (arrowhead) supplied by the anterior spinal artery (2) arising from the anterior radiculo-medullary artery (1) and draining into the cranium along the ventral surface of spinal cord (3). C) Superselective injection via the anterior spinal artery (2) with Magic microcatheter, A-P view, shows the varix (arrowhead) and the draining vein (3). D) Plain film after IBCA embolisation shows IBCA deposited within the varix and the terminal portion of the feeder (arrows). E) Selective injection of the left T4 intercostal artery after embolisation, A-P view, shows complete obliteration of the varix and draining vein. Unfortunately, the patient did not improve. F) Artist's drawing of the AVF's angioarchitecture (anterior view).

Comment – *In this case, the patient did not improve after embolisation. The possible reason is that the spinal cord was injured by the previous surgery. Microcoils are recommended to occlude the fistula so that the anterior spinal artery can be preserved.*

Section 3
Spinal Dural Arteriovenous Fistulas

Spinal dural arteriovenous fistula (SDAVF) is defined as a spinal vascular lesion, a small fistula-dominated vascular nidus or a direct arteriovenous shunt is within the spinal dura in the vicinity of the nerve root, supplied by small dural arterioles with medullary venous drainage. Because of the direct communication between the artery and the medullary vein, hypertension of the medullary vein will impede the normal venous drainage of the spinal cord and subsequently lead to congestion, oedema, even cord necrosis.

SDAVFs frequently occur in middle-aged and elderly adults. There is an male predominance with a 7.8:1 male to female ratio. Only five cases are female in our series of 39 cases. The most common clinical presentations are progressive ascending paraparesis and numbness followed by bladder and bowel dysfunction. No subarachnoid haemorrhage occurred in any patient. Clinically these lesions are often misdiagnosed as sciatica or arachnoiditis. Most patients with SDAVFs will develop irreversible paraplegia four years after the first symptom appears. So early diagnosis and treatment are very important.

All of SDAVFs were located in the thoracic, lumbar, and sacral areas. No cervical SDAVF was encountered. These lesions have their own characteristic features in MRI and angiography. 50% of the patients can be curatively embolised. If the microcatheter cannot reach the fistula, surgical clipping is the alternative.

In our series of 39 patients with SDAVFs, eight were located above the T8 level, 31 below this level. Nine cases were treated by embolisation alone, 24 by surgery alone, and four cases were treated with a combination of embolisation and surgery, and two cases left untreated. As concerns the therapeutic outcome, 21 cases were excellent, nine cases were good, six cases fair, two cases no change, one worse.

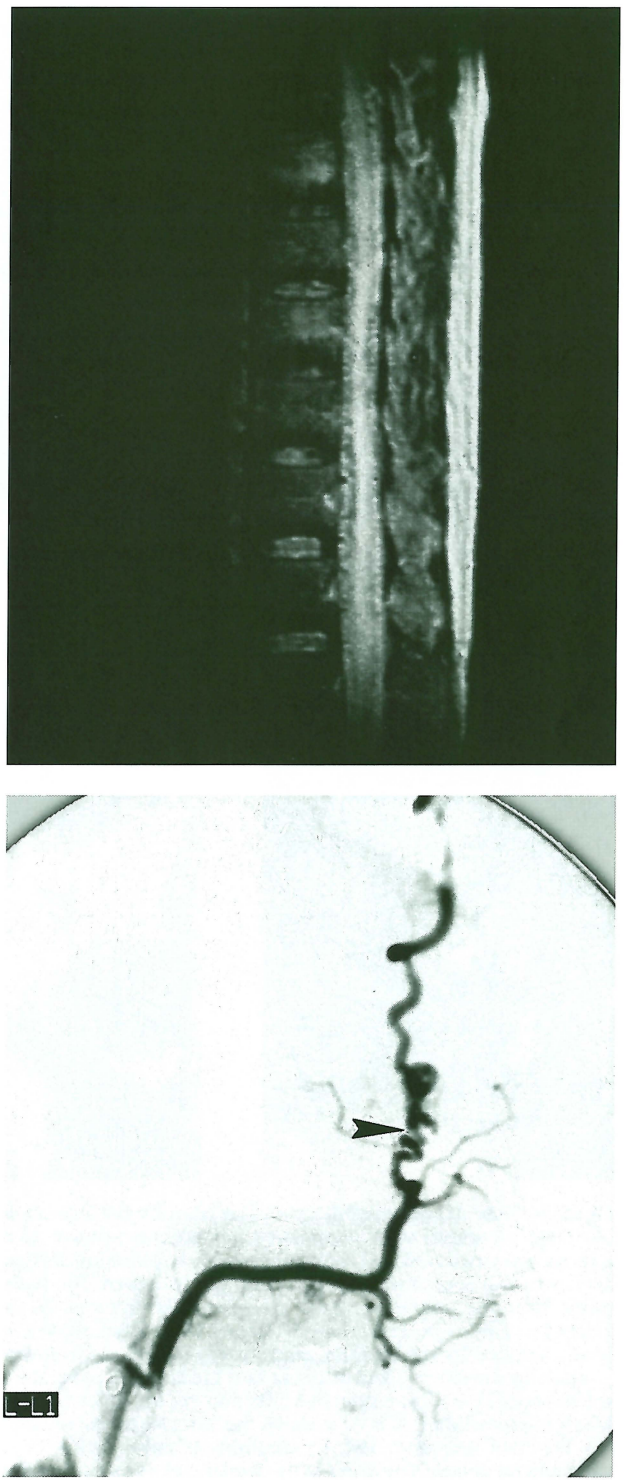


Figure 7 SDAVF treated by NBCA embolisation. A 65-year-old male presented with progressive paraparesis for one year. A) A sagittal T2-weighted MR image shows the hyperintensity signal in the thoracic cord which is related to oedema. B) Selective angiography of the left L1 lumbar artery in lateral view shows the fistula (arrowhead) supplied by a dilated dural artery and draining to the medullary vein.

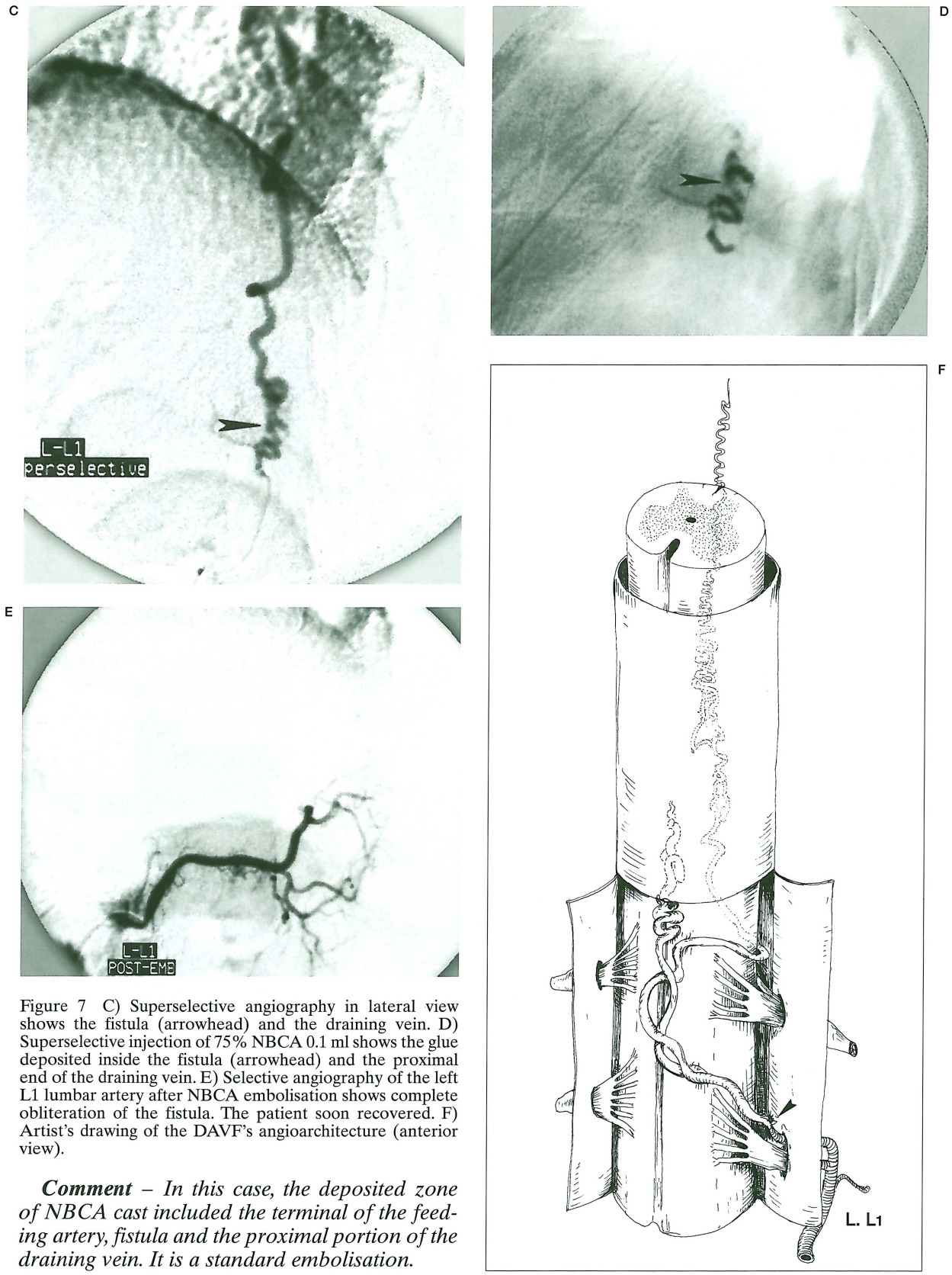
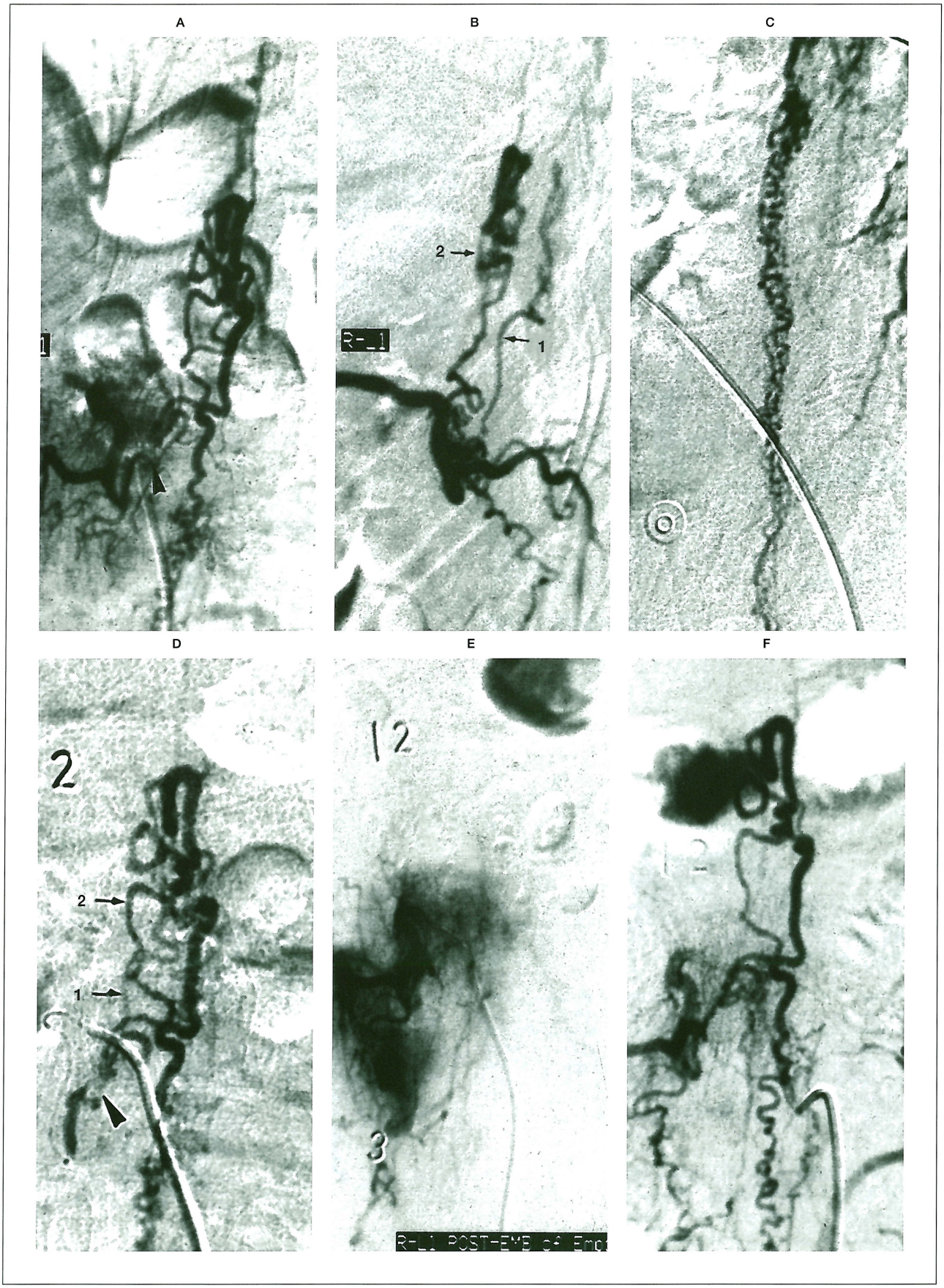


Figure 7 C) Superselective angiography in lateral view shows the fistula (arrowhead) and the draining vein. D) Superselective injection of 75% NBCA 0.1 ml shows the glue deposited inside the fistula (arrowhead) and the proximal end of the draining vein. E) Selective angiography of the left L1 lumbar artery after NBCA embolisation shows complete obliteration of the fistula. The patient soon recovered. F) Artist's drawing of the DAVF's angioarchitecture (anterior view).

Comment *– In this case, the deposited zone of NBCA cast included the terminal of the feeding artery, fistula and the proximal portion of the draining vein. It is a standard embolisation.*


A
B
C
2
1
R-_1
D
E
F
2
2
1
12
3
12

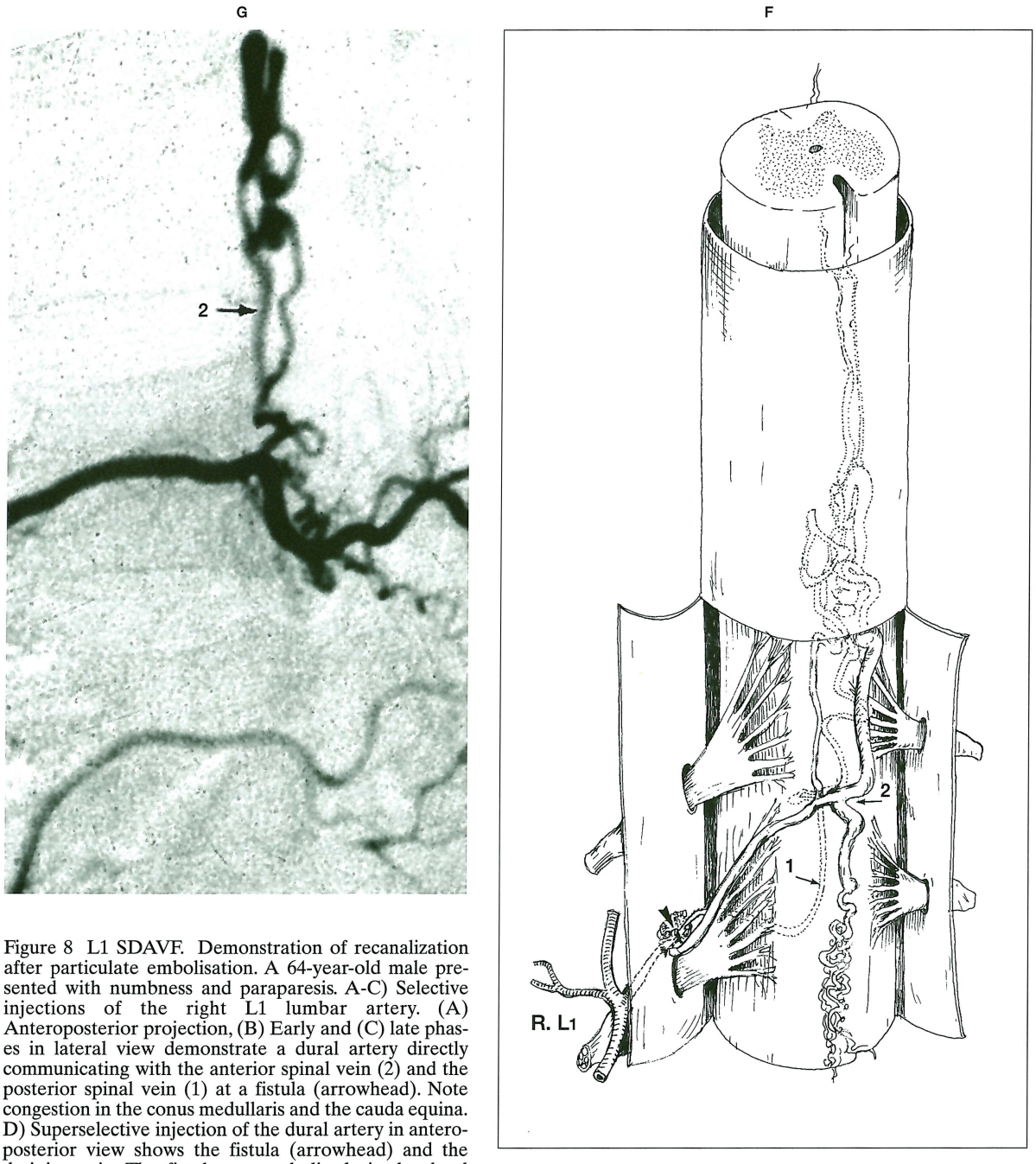


Figure 8 L1 SDAVF. Demonstration of recanalization after particulate embolisation. A 64-year-old male presented with numbness and paraparesis. A-C) Selective injections of the right L1 lumbar artery. (A) Anteroposterior projection, (B) Early and (C) late phases in lateral view demonstrate a dural artery directly communicating with the anterior spinal vein (2) and the posterior spinal vein (1) at a fistula (arrowhead). Note congestion in the conus medullaris and the cauda equina. D) Superselective injection of the dural artery in anteroposterior view shows the fistula (arrowhead) and the draining vein. The fistula was embolised via the dural artery with Embosphere 900 micron in diameter. E) Post-embolisation angiogram shows a disappearance of the fistula. F-C) Follow-up arteriograms of the right L1 lumbar artery one year after embolisation in (F) anteroposterior and (G) lateral projections show recanalization of the fistula and the anterior draining vein (2). The posterior spinal vein is not opacified this time. H) Artist's drawing of the DAVF angioarchitecture (anterior view).

Comment – *Through the treatment of this case, it was further verified that particulate embolisation is only temporary. Because the lesion was misdiagnosed as a intramedullary AVM. Embosphere was used at that time. Intensive angiographic study after recanalization proved it to be a dural AVF, which was then treated by surgery.*

EDITORIAL COMMENT

We are pleased to publish this Chinese Experience in Endovascular Management of Spinal Cord Vascular Malformations despite the fact that it is part of a textbook and does not follow the rules of publication that have been recommended. This important series studied in three different Chinese centres is one of the largest available in the literature. The number of patients treated, as well as the immediate results obtained by this team, deserved this exception.

A selection from the large iconography initially proposed in the author's textbook has been made. The pictures that are shown reflect not only the variety of pathologies that the authors had to tackle, but also the different managements they proposed.

Good clinical results have been obtained and the immediate anatomical results that are shown seem satisfactory. One can note the absence of long-term follow-up in most of the cases this paper proposes. It is therefore open to another discussion far beyond the technical approaches that are described, as it is largely accepted that most of these spinal cord lesions often cannot be cured.

One must therefore escape the images of the disease detected by MRI or angiography in order to focus the analysis on the couple formed by the AVM and the surrounding spinal cord tissue. The richness of the intrinsic and extrinsic vascular network of the spinal cord makes the understanding of the architecture of the malformation, and by extension the treatment that should be applied, often hazardous and difficult. The "wedged catheter position" that is advocated by many for dural or brain arteriovenous shunts to obtain an optimal disconnection of the nidus, should not be performed in spinal cord lesions. Complications can be due to contamination by glue or particles of enlarged but normal intrinsic vessels (angioectatic), ignored or erroneously considered pathological. If glue is used as the best embolic material for spinal cord arteriovenous malformations, Histoacryl injected in a wedged position can cast a spinal cord AVM optimally but it may produce a triple injury to the nervous tissue or intrinsic vasculature: thermic, thrombotic, or inflammatory.

Balloons are often difficult to navigate in spinal cord arteries because of their tortuosity. Coils can sometimes be detached in venous ectasias, but the catheters necessary for the delivery of these coils are often too stiff to pass all the curves made by these vessels and can only be used in certain particular anatomical circumstances with rather straight vessels. Particles may produce total thrombosis (confirmed at long term follow-up examinations) in very selected small-sized slow flow lesions. Particles should not be used as the primary agent as they may (as seen in one illustrative example of this paper) just occlude the shunt temporarily. Secondary development of other feeding arteries, hindering any further satisfactory more selective approach, complicates the architecture secondarily and requires poorly efficient repetitive endovascular sessions.

The main tools available for treatment of spinal cord arteriovenous malformations are poorly adapted to obtain a cure in many of these lesions. It seems more acceptable to look for a long-term clinical improvement or at least stabilization. As for brain AVMs, and even more in spinal cord AVMs, partial targetted endovascular therapy with NBCA has a positive effect on the natural history of cord AVMs. Further assessments and controls have naturally to be made to confirm these observations and experience. Research should be focused not only on the "phenotypic aspects" of these lesions but it should aim to shed more light on the biology and interactions between the shunt and the surrounding nervous tissue.

Dr Feng Ling and her co-workers have to be congratulated for their large series, the excellent results obtained and the inventiveness they have developed in order to manage and approach these difficult malformations satisfactorily.

Georges Rodesch M.D.
Hôpital Bicêtre, Université de Paris Sud